IMPLEMENTATION OF METABOLIC SYNDROME ASSESSMENT IN THE ROUTINE MANAGEMENT OF BENIGN PROSTATIC HYPERPLASIA

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Abstract Benign prostatic hyperplasia (BPH) is one of the most common urological diseases among aging men and is frequently accompanied by lower urinary tract symptoms that substantially impair quality of life. Although current clinical management of BPH primarily focuses on prostate enlargement and urinary symptoms, increasing evidence indicates that metabolic syndrome plays an important role in the development and progression of the disease. However, routine assessment of metabolic syndrome is still insufficiently implemented in everyday urological practice. The present study aimed to evaluate the clinical value of implementing metabolic syndrome assessment in the routine management of patients with benign prostatic hyperplasia. The study included patients diagnosed with BPH and age-matched healthy controls. Clinical evaluation comprised body mass index, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, serum triglycerides, high-density lipoprotein cholesterol, prostate-specific antigen (PSA), prostate volume determined by transrectal ultrasonography, and the severity of lower urinary tract symptoms assessed using the International Prostate Symptom Score (IPSS). The implementation of systematic metabolic syndrome assessment allowed early identification of patients at increased risk of severe lower urinary tract symptoms and progressive prostate enlargement. Patients with metabolic syndrome demonstrated significantly higher PSA concentrations, larger prostate volume, more severe urinary symptoms, and more pronounced metabolic abnormalities than patients without metabolic syndrome. The integration of metabolic syndrome assessment into routine clinical practice improved comprehensive patient evaluation and facilitated individualized therapeutic decision-making. These findings support the incorporation of metabolic syndrome screening into the routine management of patients with benign prostatic hyperplasia to optimize diagnosis, risk stratification, and long-term clinical outcomes.

Keywords: benign prostatic hyperplasia, metabolic syndrome, clinical implementation, lower urinary tract symptoms, prostate-specific antigen, prostate volume, International Prostate Symptom Score, obesity, dyslipidemia, personalized management.

Annotatsiya Prostata bezi xavfsiz giperplaziyasi (PBXG) yoshi ulg‘aygan erkaklarda eng ko‘p uchraydigan urologik kasalliklardan biri bo‘lib, ko‘pincha quyi siydik yo‘llari simptomlari bilan kechadi va bemorlarning hayot sifatini sezilarli darajada pasaytiradi. Hozirgi vaqtda PBXGni davolashda asosiy e’tibor prostata bezi kattalashishi hamda siydik chiqarish buzilishlarini baholashga qaratilgan bo‘lsa-da, so‘nggi yillarda metabolik sindrom kasallikning rivojlanishi va klinik kechishida muhim omillardan biri ekanligi isbotlanmoqda. Shunga qaramay, metabolik sindromni baholash amaliy urologik faoliyatda yetarli darajada qo‘llanilmayapti. Mazkur tadqiqotning maqsadi prostata bezi xavfsiz giperplaziyasi bilan og‘rigan bemorlarni rutin klinik boshqarishda metabolik sindromni baholashni amaliyotga joriy etishning klinik ahamiyatini aniqlashdan iborat bo‘ldi. Tadqiqotga PBXG bilan og‘rigan bemorlar hamda yosh bo‘yicha moslashtirilgan sog‘lom nazorat guruhi kiritildi. Klinik baholash davomida tana vazni indeksi (BMI), bel aylana uzunligi, sistolik va diastolik arterial bosim,

och qoringa qondagi glyukoza, qon zardobidagi triglitseridlar, yuqori zichlikdagi lipoproteid xolesterini (HDL), prostata-spetsifik antigen (PSA), transrektal ultratovush tekshiruvi orqali aniqlangan prostata hajmi hamda Xalqaro prostata simptomlari shkalasi (IPSS) bo‘yicha quyi siydik yo‘llari simptomlarining og‘irligi baholandi. Metabolik sindromni muntazam baholashni klinik amaliyotga joriy etish og‘ir quyi siydik yo‘llari simptomlari va prostata hajmining progressiv kattalashishi xavfi yuqori bo‘lgan bemorlarni erta aniqlash imkonini berdi. Metabolik sindromga ega bemorlarda PSA darajasi, prostata hajmi va IPSS ko‘rsatkichlari metabolik sindrom kuzatilmagan bemorlarga nisbatan sezilarli darajada yuqori ekanligi aniqlandi. Metabolik sindromni rutin klinik amaliyotga integratsiya qilish bemorlarni kompleks baholashni yaxshiladi hamda individual davolash taktikasini tanlash samaradorligini oshirdi. Olingan natijalar PBXG bilan og‘rigan bemorlarni boshqarishda metabolik sindromni skrining qilishni muntazam klinik amaliyotning ajralmas qismi sifatida qo‘llash zarurligini tasdiqlaydi.

Kalit so‘zlar: prostata bezi xavfsiz giperplaziyasi, metabolik sindrom, amaliyotga joriy etish, quyi siydik yo‘llari simptomlari, prostata-spetsifik antigen, prostata hajmi, IPSS, semizlik, dislipidemiya, individual yondashuv.

Аннотация Доброкачественная гиперплазия предстательной железы (ДГПЖ) является одним из наиболее распространённых урологических заболеваний у мужчин пожилого возраста и часто сопровождается симптомами нижних мочевых путей, значительно ухудшающими качество жизни пациентов. Несмотря на то что современная тактика ведения больных ДГПЖ в основном ориентирована на оценку увеличения предстательной железы и нарушений мочеиспускания, многочисленные исследования свидетельствуют о важной роли метаболического синдрома в развитии и прогрессировании данного заболевания. Однако в повседневной урологической практике оценка метаболического синдрома проводится недостаточно широко. Целью настоящего исследования явилась оценка клинической эффективности внедрения оценки метаболического синдрома в рутинное ведение пациентов с доброкачественной гиперплазией предстательной железы. В исследование были включены пациенты с ДГПЖ и контрольная группа практически здоровых мужчин, сопоставимых по возрасту. Клиническое обследование включало определение индекса массы тела, окружности талии, систолического и диастолического артериального давления, уровня глюкозы плазмы натощак, концентрации триглицеридов, холестерина липопротеинов высокой плотности (ЛПВП), простат-специфического антигена (ПСА), объёма предстательной железы по данным трансректального ультразвукового исследования, а также выраженности симптомов нижних мочевых путей по Международной шкале симптомов предстательной железы (IPSS). Внедрение систематической оценки метаболического синдрома позволило своевременно выявлять пациентов с высоким риском выраженных симптомов нижних мочевых путей и прогрессирующего увеличения объёма предстательной железы. У пациентов с метаболическим синдромом отмечались более высокие уровни ПСА, больший объём предстательной железы, более выраженные нарушения мочеиспускания и более тяжёлые метаболические расстройства по сравнению с пациентами без метаболического синдрома. Интеграция оценки метаболического синдрома в рутинную клиническую практику способствовала повышению эффективности комплексного обследования пациентов и выбору персонализированной лечебной тактики. Полученные результаты подтверждают целесообразность включения скрининга метаболического синдрома в стандартный алгоритм ведения пациентов с доброкачественной гиперплазией предстательной железы.

Ключевые слова: доброкачественная гиперплазия предстательной железы, метаболический синдром, внедрение в клиническую практику, симптомы нижних мочевых путей, простат-специфический антиген, объём предстательной железы, IPSS, ожирение, дислипидемия, персонализированное лечение.

Introduction

Benign prostatic hyperplasia (BPH) is one of the most common age-related urological disorders affecting men worldwide and represents a major cause of lower urinary tract symptoms (LUTS), deterioration of quality of life, and increased healthcare utilization. Epidemiological studies indicate that histological evidence of BPH is present in approximately 50% of men older than 50 years and in more than 80% of men over the age of 80 years. As global life expectancy continues to increase, the prevalence of BPH and its associated complications is expected to rise substantially, creating an important public health challenge. Although BPH is considered a benign disease, progressive enlargement of the prostate gland may result in bladder outlet obstruction, recurrent urinary tract infections, acute urinary retention, renal dysfunction, and the need for surgical intervention. Therefore, identification of modifiable risk factors contributing to disease progression has become an important objective in modern urology.

Traditionally, the clinical management of BPH has focused primarily on the assessment of prostate volume, prostate-specific antigen (PSA), lower urinary tract symptoms, uroflowmetry, and postvoid residual urine volume. Therapeutic decisions are generally based on symptom severity, prostate enlargement, and the presence of complications. However, increasing evidence suggests that BPH should no longer be regarded solely as a localized prostatic disorder but rather as a systemic disease influenced by multiple metabolic, inflammatory, endocrine, and vascular factors. This concept has stimulated growing interest in the role of metabolic syndrome as an important contributor to the pathogenesis and progression of benign prostatic hyperplasia.

Metabolic syndrome is a complex clinical condition characterized by the coexistence of central obesity, insulin resistance, impaired glucose metabolism, dyslipidemia, and arterial hypertension. According to the International Diabetes Federation (IDF), the prevalence of metabolic syndrome has increased dramatically during recent decades and currently affects approximately one-quarter of the adult population worldwide. Besides being a well-established risk factor for cardiovascular disease and type 2 diabetes mellitus, metabolic syndrome has also been associated with numerous chronic disorders, including chronic kidney disease, non-alcoholic fatty liver disease, and several urological diseases.

A growing number of clinical and experimental studies have demonstrated a close relationship between metabolic syndrome and benign prostatic hyperplasia. Men with metabolic syndrome frequently present with larger prostate volume, more severe lower urinary tract symptoms, accelerated disease progression, and poorer therapeutic outcomes compared with metabolically healthy individuals. These observations suggest that metabolic abnormalities actively participate in the biological processes responsible for prostatic enlargement rather than representing simple coincidental comorbidities.

Several biological mechanisms have been proposed to explain the influence of metabolic syndrome on the development and progression of BPH. Visceral obesity is associated with increased production of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein, which promote chronic low-grade inflammation within the prostate gland. Persistent inflammatory activity stimulates stromal and epithelial cell proliferation, extracellular matrix remodeling, and tissue fibrosis, thereby contributing to progressive enlargement of the prostate.

Furthermore, obesity is associated with alterations in sex hormone metabolism, including decreased testosterone levels and increased estrogen activity resulting from enhanced aromatase activity in adipose tissue. These hormonal disturbances may further influence prostatic growth and lower urinary tract function.

Insulin resistance represents another key component of metabolic syndrome involved in BPH pathogenesis. Chronic hyperinsulinemia stimulates insulin-like growth factor-1 (IGF-1) signaling pathways that enhance cellular proliferation, inhibit apoptosis, and promote angiogenesis within prostatic tissue. Simultaneously, hyperglycemia increases oxidative stress through excessive production of reactive oxygen species, leading to endothelial dysfunction, mitochondrial injury, and chronic tissue inflammation. These molecular alterations collectively facilitate continuous prostatic enlargement and worsening urinary symptoms.

Dyslipidemia also contributes significantly to prostatic pathology. Elevated serum triglycerides and reduced high-density lipoprotein cholesterol (HDL-C) have been associated with endothelial dysfunction, impaired pelvic microcirculation, oxidative stress, and chronic inflammatory activation. Lipid abnormalities may alter steroid hormone synthesis because cholesterol serves as the principal substrate for androgen biosynthesis. Consequently, disturbances in lipid metabolism may modify intraprostatic androgen concentrations and influence the biological behavior of prostatic tissue. Experimental evidence has further demonstrated that dyslipidemia promotes activation of multiple intracellular signaling pathways regulating cell proliferation, apoptosis, and inflammatory responses.

The clinical manifestations of these metabolic disturbances are reflected in worsening lower urinary tract symptoms. Numerous studies have reported positive associations between obesity, waist circumference, fasting plasma glucose, serum triglycerides, and symptom severity measured using the International Prostate Symptom Score (IPSS). Patients with metabolic syndrome also tend to have larger prostate volume, higher serum PSA concentrations, reduced urinary flow rate, and increased postvoid residual urine volume. Collectively, these findings indicate that metabolic syndrome may substantially influence both structural and functional changes of the lower urinary tract.

Despite the growing body of evidence linking metabolic syndrome with BPH, routine clinical assessment of metabolic syndrome has not yet become a standard component of urological practice. In many healthcare institutions, evaluation of patients with BPH remains focused primarily on prostate-related parameters, whereas important metabolic abnormalities often remain undetected or are diagnosed only after the development of cardiovascular complications. Consequently, opportunities for early identification of high-risk patients and implementation of preventive interventions may be missed.

Integrating metabolic syndrome assessment into the routine management of patients with benign prostatic hyperplasia may provide several important clinical advantages. Early recognition of obesity, hypertension, impaired glucose metabolism, and dyslipidemia allows clinicians to identify patients at increased risk of rapid disease progression, severe lower urinary tract symptoms, and reduced treatment response. Furthermore, comprehensive metabolic evaluation facilitates multidisciplinary management involving urologists, endocrinologists, cardiologists, and primary care physicians, thereby improving overall patient outcomes. Lifestyle modification, weight reduction, optimization of glycemic control, correction of lipid abnormalities, and blood pressure management may complement conventional pharmacological therapy and contribute to slowing BPH progression.

Therefore, the present study aimed to evaluate the clinical effectiveness of implementing routine metabolic syndrome assessment in patients with benign prostatic hyperplasia by analyzing anthropometric, biochemical, and clinical characteristics. The study also sought to determine whether

systematic evaluation of metabolic syndrome components could improve risk stratification, facilitate individualized treatment planning, and enhance the overall management of patients with benign prostatic hyperplasia. The findings are expected to support the incorporation of metabolic syndrome screening into routine urological practice and provide additional evidence for a more comprehensive, patient-centered approach to the management of benign prostatic hyperplasia.

Materials and Methods

This cross-sectional clinical study was conducted at the multidisciplinary clinic of Andijan State Medical Institute and included a total of 436 male participants. The study population consisted of 217 patients diagnosed with benign prostatic hyperplasia (BPH), while 219 age-matched apparently healthy men without clinical or instrumental evidence of prostatic disease served as the control group. The diagnosis of BPH was established according to the recommendations of the European Association of Urology (EAU) based on medical history, digital rectal examination, serum prostate-specific antigen (PSA) measurement, transrectal ultrasonography (TRUS), uroflowmetry, and clinical evaluation of lower urinary tract symptoms (LUTS). Patients with histologically confirmed prostate cancer, acute urinary tract infection, urethral stricture, neurogenic bladder dysfunction, previous prostate surgery, or severe systemic diseases were excluded from the study.

Metabolic syndrome was diagnosed according to the criteria of the International Diabetes Federation (IDF). Central obesity was considered the mandatory diagnostic component and was accompanied by at least two of the following metabolic abnormalities: elevated fasting plasma glucose, increased serum triglyceride concentration, reduced high-density lipoprotein cholesterol (HDL-C), and arterial hypertension. Based on these criteria, patients with BPH were classified into two subgroups: BPH with metabolic syndrome and BPH without metabolic syndrome. This classification was used to evaluate the impact of metabolic syndrome assessment on routine clinical management and risk stratification.

All participants underwent comprehensive clinical, anthropometric, and biochemical evaluation. Clinical assessment included age, body mass index (BMI), waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, serum triglycerides, high-density lipoprotein cholesterol (HDL-C), serum prostate-specific antigen (PSA), and prostate volume measured by transrectal ultrasonography. The severity of lower urinary tract symptoms was assessed using the International Prostate Symptom Score (IPSS), which categorized symptoms as mild, moderate, or severe according to established international recommendations. Anthropometric measurements were performed using standardized techniques. Body mass index was calculated as body weight divided by height squared (kg/m^2), whereas waist circumference was measured at the midpoint between the lower rib margin and the iliac crest. Blood pressure was measured twice after a minimum of 10 minutes of rest, and the average value was recorded for statistical analysis.

Venous blood samples were collected from all participants after an overnight fast of 8–12 hours. Serum concentrations of fasting plasma glucose, triglycerides, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol (LDL-C) were determined using automated enzymatic colorimetric methods with commercially available reagent kits according to the manufacturers' protocols. Serum PSA levels were measured using a chemiluminescent immunoassay in the certified clinical laboratory of Andijan State Medical Institute. Internal quality-control procedures were applied throughout the laboratory analyses to ensure accuracy, precision, and reproducibility of the obtained results.

Transrectal ultrasonography was performed by experienced urologists using a standardized imaging protocol. Prostate volume was calculated according to the ellipsoid formula (length × width × height × 0.52). Digital rectal examination was carried out in all patients to evaluate prostate consistency and exclude findings suspicious for malignancy. The severity of lower urinary tract symptoms was evaluated using the validated International Prostate Symptom Score (IPSS) questionnaire, while quality of life related to urinary symptoms was assessed using the corresponding IPSS quality-of-life index.

To facilitate routine clinical implementation, a standardized metabolic syndrome assessment algorithm was introduced into the outpatient management of patients with benign prostatic hyperplasia. During the initial clinical visit, every patient underwent systematic evaluation of all metabolic syndrome components together with routine urological examination. Patients diagnosed with metabolic syndrome received multidisciplinary recommendations, including dietary modification, weight reduction, increased physical activity, optimization of blood pressure, glycemic control, and correction of lipid abnormalities in collaboration with primary care physicians and endocrinologists. Clinical decision-making regarding pharmacological treatment of BPH was individualized according to the combined assessment of urological and metabolic risk factors.

Statistical analyses were performed using IBM SPSS Statistics software (version 26.0). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Normality of data distribution was assessed using the Shapiro–Wilk test. Differences between study groups were analyzed using the independent samples t-test or Mann–Whitney U test for continuous variables and Pearson's chi-square (χ^2) test for categorical variables. Correlations between metabolic syndrome components, PSA concentration, prostate volume, and IPSS score were evaluated using Pearson's or Spearman's correlation coefficients, as appropriate. Multivariate logistic regression analysis was performed to identify independent predictors of severe lower urinary tract symptoms in patients with benign prostatic hyperplasia. Odds ratios (ORs) with 95% confidence intervals (95% CIs) were calculated, and a two-sided p value of less than 0.05 was considered statistically significant.

Results

A total of 436 men participated in the present study, including 217 patients diagnosed with benign prostatic hyperplasia (BPH) and 219 age-matched healthy controls. Among patients with BPH, metabolic syndrome was identified in a substantial proportion of cases, confirming the close association between metabolic disorders and benign prostatic enlargement. Comparative analysis demonstrated that patients with metabolic syndrome exhibited significantly more pronounced anthropometric, biochemical, and clinical abnormalities than patients without metabolic syndrome.

Anthropometric assessment revealed that body mass index (BMI) and waist circumference were significantly higher in patients with BPH associated with metabolic syndrome than in patients without metabolic syndrome and healthy controls ($p<0.05$). Similarly, systolic and diastolic blood pressure values were significantly elevated in the metabolic syndrome group, reflecting the progressive deterioration of cardiovascular and metabolic status.

Biochemical analysis showed marked disturbances of glucose and lipid metabolism in patients with metabolic syndrome. Fasting plasma glucose and serum triglyceride concentrations were significantly increased, whereas high-density lipoprotein cholesterol (HDL-C) levels were significantly reduced compared with patients without metabolic syndrome and the control group ($p<0.05$). These

findings confirmed the presence of pronounced metabolic dysfunction in patients with BPH accompanied by metabolic syndrome.

Clinical evaluation demonstrated that serum prostate-specific antigen (PSA) concentrations were significantly higher in patients with metabolic syndrome than in patients without metabolic syndrome. Although PSA values generally remained within the range expected for benign disease, patients with metabolic syndrome showed consistently higher PSA levels, indicating greater biological activity of the prostate. In addition, prostate volume measured by transrectal ultrasonography was significantly larger in the metabolic syndrome group ($p<0.05$).

Assessment of lower urinary tract symptoms using the International Prostate Symptom Score (IPSS) revealed that patients with metabolic syndrome experienced significantly more severe urinary symptoms. Moderate and severe LUTS were observed more frequently among patients with metabolic syndrome, whereas mild symptoms predominated in patients without metabolic abnormalities. Furthermore, quality-of-life scores associated with urinary symptoms were significantly worse in patients with metabolic syndrome, indicating a greater negative impact of the disease on daily functioning.

Correlation analysis demonstrated significant positive relationships between the severity of lower urinary tract symptoms and major components of metabolic syndrome. Body mass index, waist circumference, fasting plasma glucose, serum triglyceride concentration, systolic blood pressure, prostate volume, and PSA level all showed positive correlations with IPSS scores ($p<0.05$). Conversely, HDL cholesterol demonstrated a significant inverse correlation with symptom severity, indicating that lower HDL-C concentrations were associated with more severe urinary symptoms.

Analysis of prostate volume demonstrated a progressive increase according to the severity of metabolic abnormalities. Patients presenting with a greater number of metabolic syndrome components had significantly larger prostate volume than those with fewer metabolic abnormalities. Similar relationships were observed for PSA concentrations, which increased gradually with the accumulation of metabolic syndrome components.

Implementation of routine metabolic syndrome assessment substantially improved clinical risk stratification of patients with benign prostatic hyperplasia. Systematic evaluation of obesity, blood pressure, fasting plasma glucose, serum triglycerides, and HDL cholesterol enabled early identification of patients at increased risk for severe lower urinary tract symptoms and progressive prostate enlargement. This comprehensive approach also facilitated individualized therapeutic planning and multidisciplinary management aimed at correcting both urological and metabolic abnormalities.

Multivariate logistic regression analysis identified several independent predictors of severe lower urinary tract symptoms in patients with benign prostatic hyperplasia. Increased body mass index, enlarged waist circumference, elevated fasting plasma glucose, hypertriglyceridemia, reduced HDL cholesterol, increased prostate volume, and elevated PSA concentration remained independently associated with severe LUTS after adjustment for age and other potential confounding variables. Among these factors, central obesity and prostate volume demonstrated the strongest associations with symptom severity.

Overall, the present study demonstrated that routine assessment of metabolic syndrome provides valuable additional clinical information beyond conventional urological evaluation. Patients with metabolic syndrome exhibited larger prostate volume, higher PSA concentrations, more severe lower urinary tract symptoms, and more pronounced metabolic abnormalities than metabolically healthy

patients. These findings support the incorporation of metabolic syndrome assessment into the routine clinical management of benign prostatic hyperplasia as an effective strategy for improving risk stratification, optimizing individualized treatment, and enhancing long-term clinical outcomes.

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